The Myosplint Implant Procedure

Kiyotaka Fukamachi, Patrick M. McCarthy, and Bradley Powell

Heart failure is increasing worldwide in part due to an aging population. In patients with heart failure, the heart often dilates to compensate for the decreased cardiac function. But the dilated chamber radius of the failing heart increases wall stress in proportion to the ventricular radius, according to Laplace's law (Fig IA). This increased wall stress induces further chamber dilation and deterioration in cardiac function. Surgical remodeling operations, including endoventricular patch plasty repair (Dor procedure) for ischemic cardiomyopathy and partial left ventriculectomy (PLV; Batista procedure) for dilated cardiomyopathy, reverse this ventricular remodeling process. PLV decreases the ventricular radius by resecting a large part of the left ventricle (LV). This procedure has had a high failure rate, however, partly because it requires major surgery on cardiopulmonary bypass and removes a large segment of otherwise viable muscle. The procedure may also worsen diastolic function even though systolic function improves.

A novel device, the Myosplint (Myocor, Inc., Maple Grove, MN), has been developed to change the shape of the LV and make the effective radius smaller without resecting viable muscle (Fig IB). We typically apply 20% stress reduction by tightening the Myosplint so that the new radius (R2) is 80% of the original radius (R1). The Myosplint improves ventricular function by reducing wall stress and increasing contractility. Compared with the amount of radius reduction, the resulting volume reduction is less. Therefore, the Myosplint is not expected to create major changes in diastolic function.

The concept of the Myosplint is illustrated in Fig II. Two people hold two different-sized sails in the same strength wind. The force required to hold the large sail is higher because of larger radius of the sail. If a post is solidly fixed in the ground in the middle of the sail, then the force required to hold the large sail would be much smaller because of the smaller effective radius.

In the dilated heart, shape change is achieved by placing three equally spaced Myosplints along a line from the base to the apex, as illustrated in Fig III. The goal is to place each of the Myosplints such that they equally bisect the LV chamber in the cross-sectional plane while avoiding damage to the valvular apparatus and coronary vessels. A variety of positions are possible, but the most common position is from the lateral wall (just medial or lateral to the anterolateral papillary muscle) to the posterior septum. This position was chosen to facilitate Myosplint insertion while avoiding injury to epicardial and intraventricular structures.

Patients diagnosed with dilated cardiomyopathy are appropriate candidates for this procedure. The LV end-diastolic diameter determined by two-dimensional echocardiography should preferably be between 6.5 and 12 cm. The New York Heart Association (NYHA) classification can be III or early class IV. Patients with sustained or uncontrolled atrial and/or ventricular arrhythmia, active infection, or acute myocardial infarction (within 30 days) are not appropriate candidates.

The Myosplint consists of an implantable transventricular tension member and two epicardial pads (Fig IVA). The 1.4-mm diameter polyethylene braided tension member is coated with expanded polytetrafluoroethylene (e-PTFE). The tension member is extremely durable, as evidenced by its freedom from structural deterioration at 200 million cycles (5 years) of accelerated life testing. This tension member is connected to a fixed epicardial pad constructed from high-performance engineering thermoplastic and covered with polyester fabric. A similar adjustable epicardial pad is threaded over and fixed to the tension member after implantation. The two pads are placed on opposite surfaces of the heart, with the load-bearing tension member passing through the LV, connecting the pads and drawing the ventricular walls toward one another.

Several surgical instruments have been specifically designed to facilitate implantation. The EpiProbe (Fig IVB) is used in combination with epicardial echocardiography (EE) to identify the desired location for Myosplint placement relative to internal and external cardiac anatomy by compressing the epicardial surface to create an endocardial indentation. The EpiTag (Fig IVB) is a marker deployed from the tip of the EpiProbe to mark the entry and exit points on the surface of the heart. The C-alignment device (C-device) is a surgical instrument used to guide the needle and stylet assembly from the entry to the exit points of the ventricle for precise delivery of the tension members (Fig IVC). The C-device has a guide tube to guide the placement of the needle and stylet assembly in one end and a hard stop to stop advancement of the needle and stylet assembly after placement through the ventricle in the other end. The needle and stylet assembly (Fig IVC), comprising a

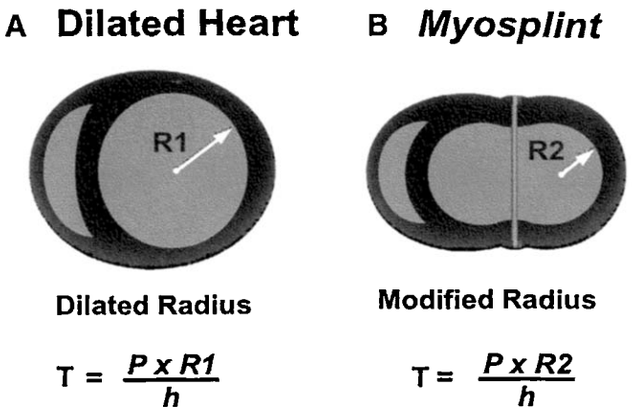


Fig I. (A) According to Laplace's law wall, stress (T) in the dilated heart is directly related to the ventricular radius (R1) and intraventricular pressure (P) and inversely related to wall thickness (h). (B) The Myosplint creates a bilobular shape with reduced wall stress due to the reduced effective radius of each lobe (R2).

needle tube and a superelastic stylet, is used with the C-device to create a pathway through the ventricle to deliver the tension member. The measurement and tightening (M/T) device is used to measure the epicardial distance of the heart and to apply the 20% stress reduction (Fig IVD). LV dimension is initially obtained by reading the position of the green index mark on the primary (black) scale of the M/T device. To apply the 20% stress reduction, the green index mark is adjusted to the same value on the second (blue) scale as is measured on the black scale.

Comments

The Myosplint improves ventricular function by reducing wall stress and increasing contractility.[1,2] Cardiac output does not increase dramatically after Myosplint implantation unlike after ventricular assist device implantation. Therefore, postoperative management is similar to that for a patient with heart failure. In addition, the administration of anticoagulation therapy (warfarin to an international normalized ratio of 2.0–3.5) for 90 days is recommended.

Other surgical approaches to treating dilated cardiomyopathy include heart transplantation, mechanical assistance devices, mitral valve repair, PLV, and dynamic cardiomyoplasty. New devices, such as Acorn's (Acorn Cardiovascular, Inc., St. Paul, MN) cardiac support device (CSD)[3] and the CardioClasp[4] (CardioClasp, Inc., Somerville, NJ) have also been developed to treat dilated cardiomyopathy.

Heart transplantation is a highly effective therapy for patients with end-stage heart failure; however, it is severely limited by the shortage of donor hearts, which is expected to never meet the overwhelming demand. Implantable left ventricular assist devices (LVADs) have demonstrated clinical success in treating patients with end-stage heart failure mainly as a bridge to transplant.[5] Recipients of these portable electrically pow-

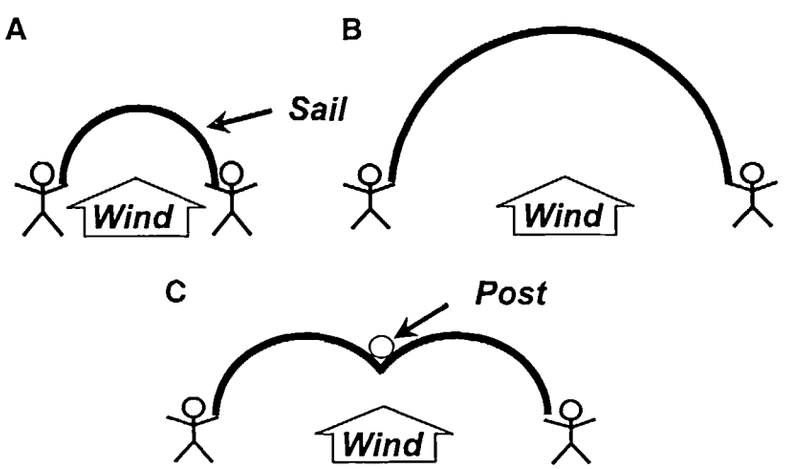


Fig II. The concept of the Myosplint can best be understood by conceiving of two people holding a sail. (A) Small sail. (B) Large sail. (C) Large sail with a post solidly fixed in the ground in the middle of the sail.

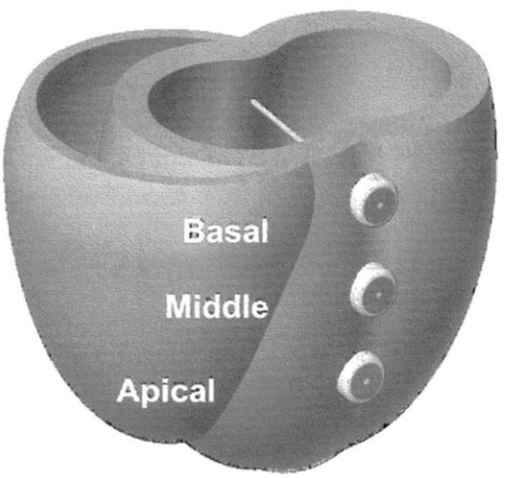


Fig III. Three transventricular Myosplints are placed along a line from the base to the apex to bisect the ventricle.

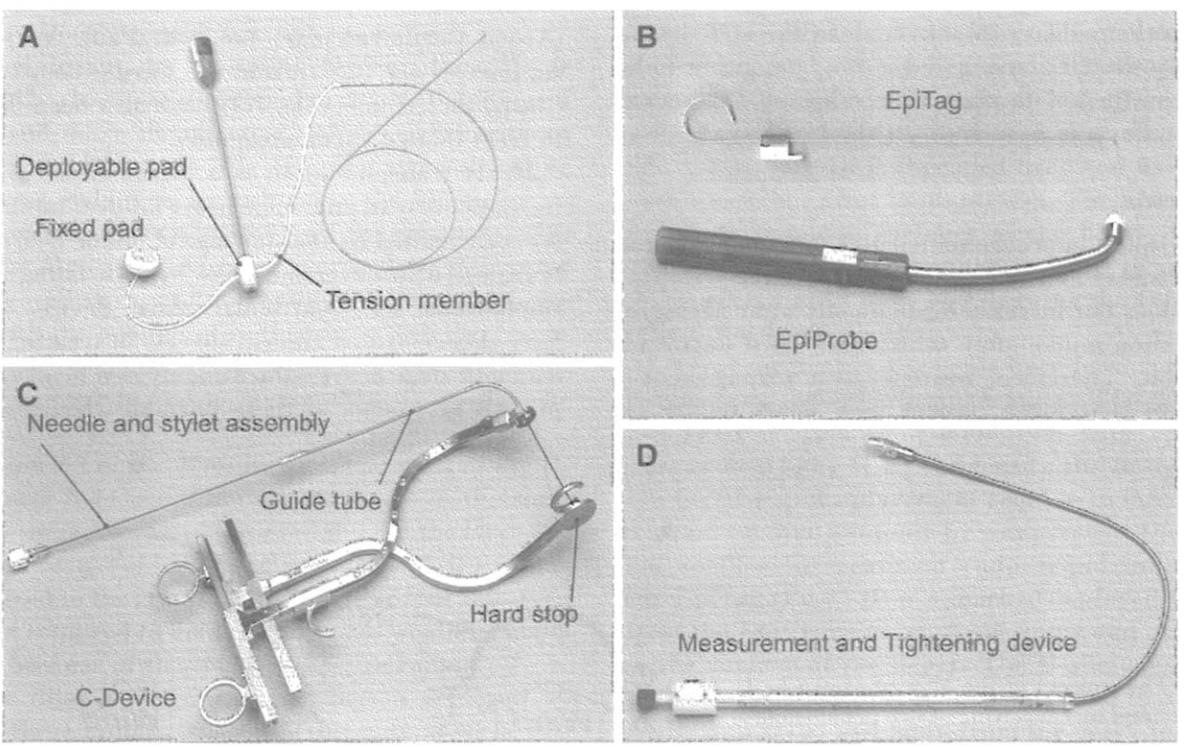
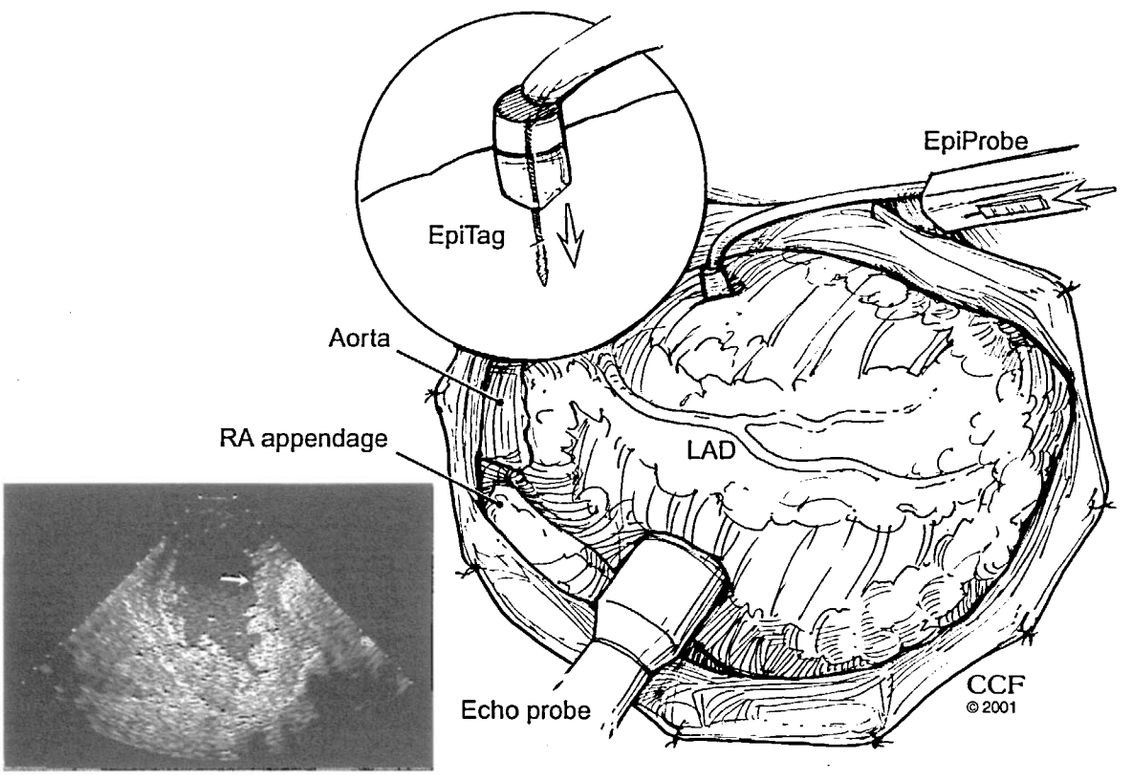


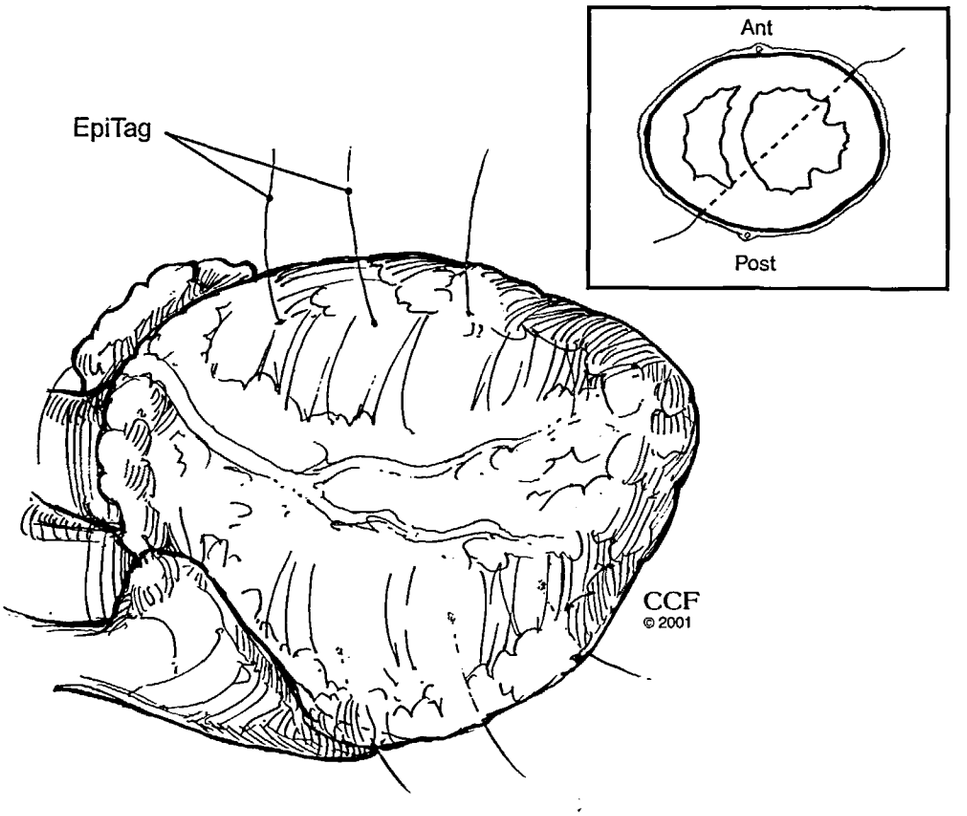
Fig IV. (A) The Myosplint (a tension member with fixed and adjustable epicardial pads). (B) The EpiProbe and EpiTag. (C) The C-device and needle and stylet assembly. (D) The M/T device.

SURGICAL TECHNIQUE

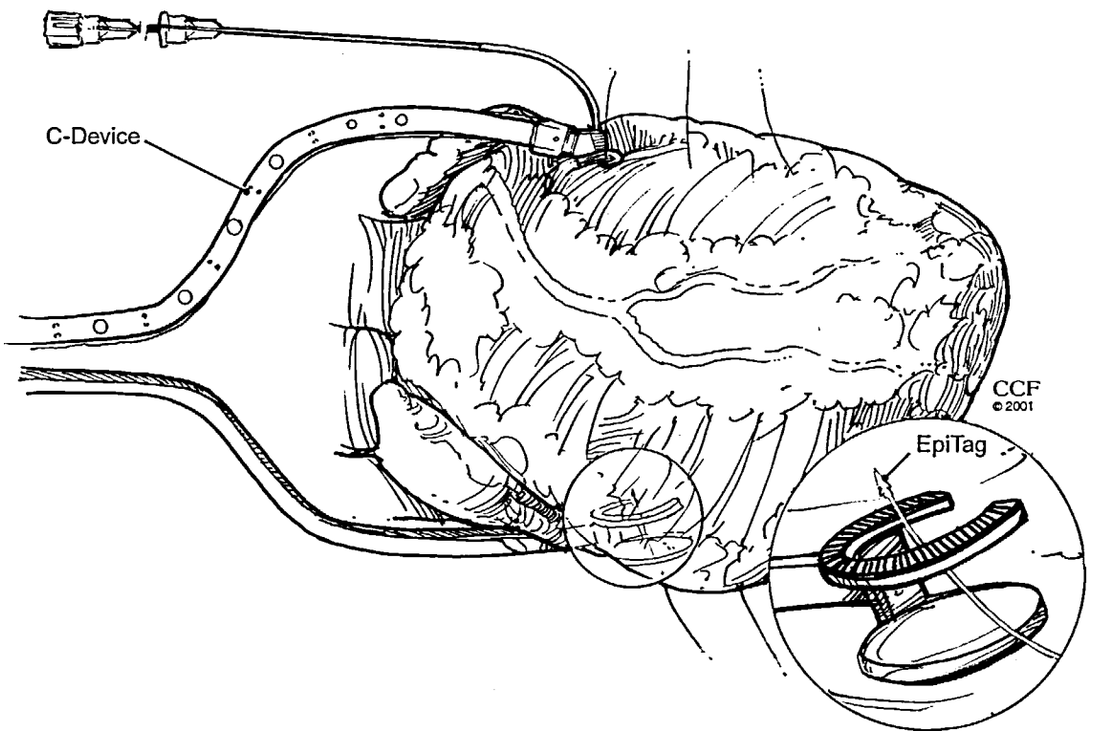


The Myosplint implantation procedure is performed through a median sternotomy incision without the use of cardiopulmonary bypass. When concomitant procedures, such as mitral valve repair, are performed, Myosplint implantation can be performed either before going on cardiopulmonary bypass or after weaning from the bypass. The implant procedure includes three distinct phases: (1) identification of positions and marking, (2) Myosplint placement, and (3) shape change process.

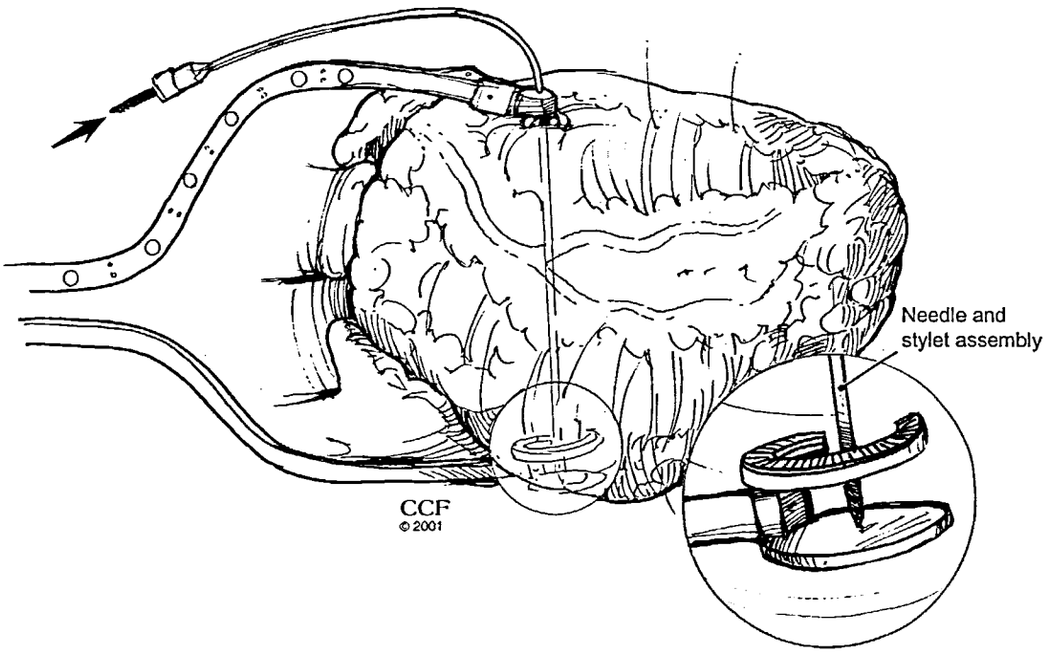
The first step is to identify the desired insertion points for the three tension members. The pericardium is lifted with pericardial stitches for better exposure of the LV lateral wall and the right ventricular (RV) posterior wall. The goal is to place each of the tension members so that they equally bisect the LV chamber in the cross-sectional plane while avoiding damage to the valvular apparatus and coronary vessels. These three tension members should be equally spaced along a line from base to the apex. First, the entry points on the LV are identified; then the corresponding exit points on the RV are identified. The entry points relative to the internal structures, such as papillary muscles, are identified by imaging with EE in the short axis. We use an EpiProbe to compress the epicardial surface and create an endocardial indentation (indicated by the arrow in the echocardiographic image) to correlate the internal anatomy and external positions of the pads. Once the position is determined, an EpiTag, which sticks on the epicardium, is deployed from the tip of the EpiProbe (inset).



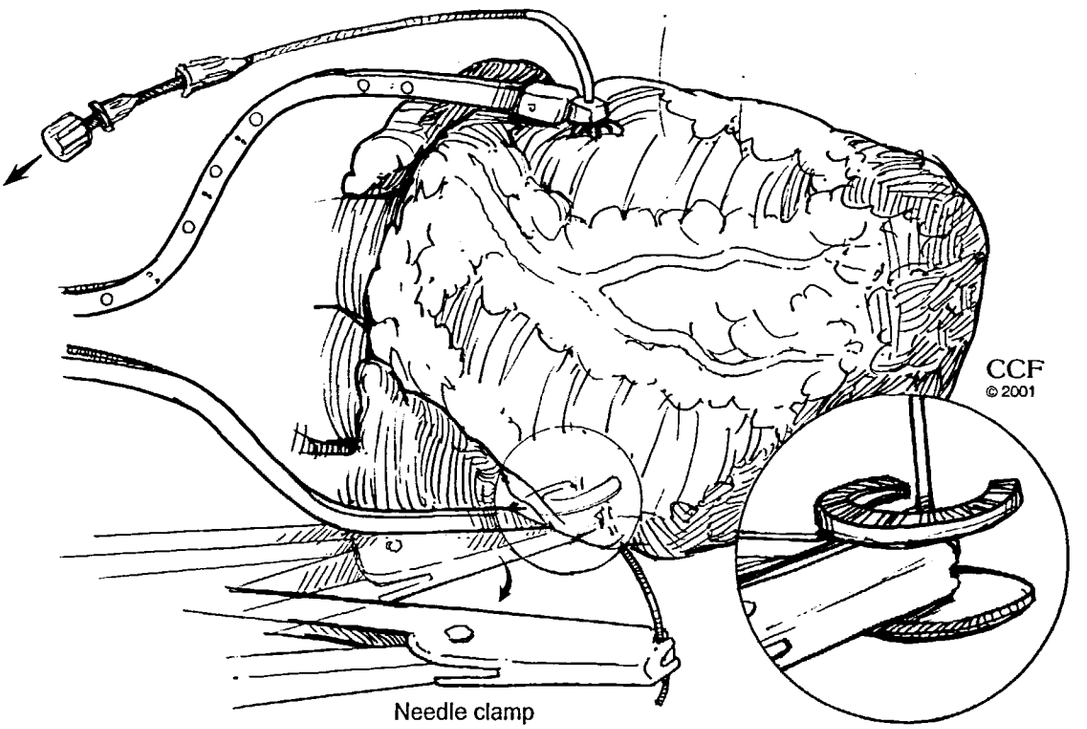
2 This illustration shows all six EpiTags in place. The first (basal) LV position is approximately 3 cm below the atrioventricular groove, and medial (or lateral) to the anterolateral papillary muscle. The second (middle) LV position is approximately 3 cm below the first position, just medial (or lateral) to the anterolateral papillary muscle. The third (apical) LV position is approximately 3 cm below the second position, placed parallel to the first two positions, running in the direction of the apex. The first (basal) RV position is approximately 3 cm below the atrioventricular groove and approximately 2 cm from the posterior descending artery at a level and an orientation that yield good bisection and avoid damage to the valvular apparatus and epicardial vessels. The second (middle) RV position is approximately 3 cm below the first position in the direction of the apex parallel to the line created by the basal positioning. The third (apical) RV position is approximately 3 cm below the second position, placed parallel to the first two positions and running in the direction of the apex.



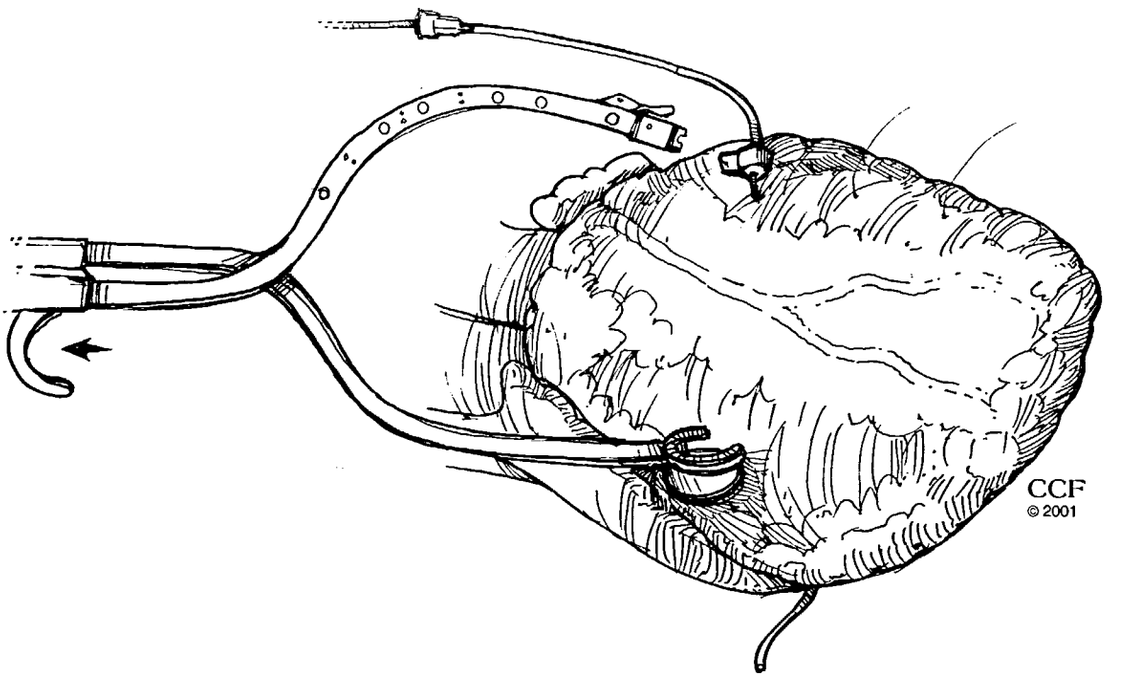
3 After all tension member locations are determined, the tension members are placed in the following order: basal, middle, and apical. A specially designed C-device is used to allow precise delivery of the tension members and to stabilize the beating heart during the delivery of a flexible needle and stylet assembly from the entry to the exit point. The needle and stylet assembly is loaded into the guide tube of the C-device. The C-device is then placed around the heart, with the tip of each end of the C-device located at the predetermined entry and exit points with EpiTags. The C-device is placed so that the side with the guide tube is placed on the LV entry point.



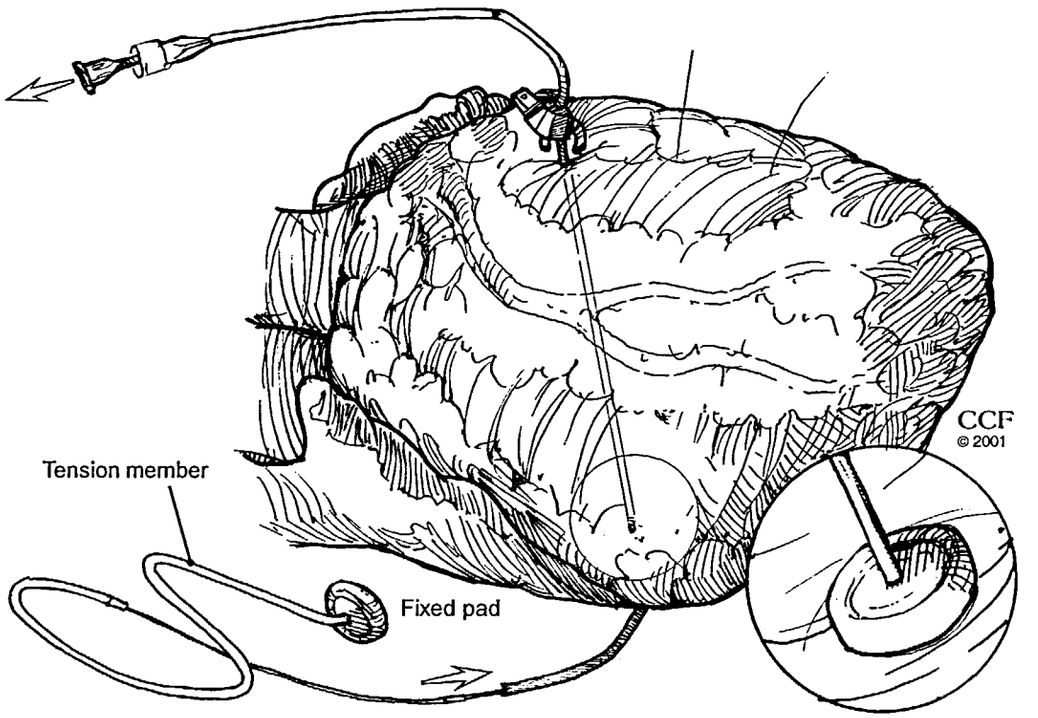
4 The needle and stylet assembly is inserted through the LV entry point, through the chamber, and out the RV until the needle hits the hard stop (see inset) of the C-device.



5 After the needle and stylet assembly is placed, the needle tube is grasped with a needle clamp and the stylet is removed from the needle tube. The flexible needle tube is pulled through the heart to allow easy access to the end of the needle tube for placing the tension member.

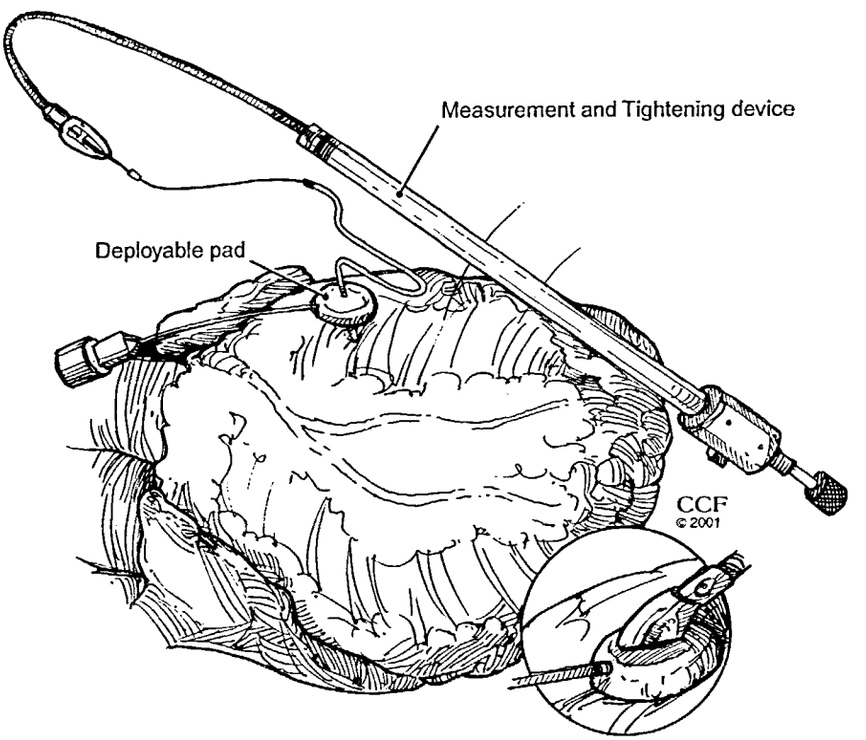


6 Once the needle tube is secured, the C-device is detached from the guide tube and removed from the chest cavity. EpiTags are removed by pulling on the attached leader.

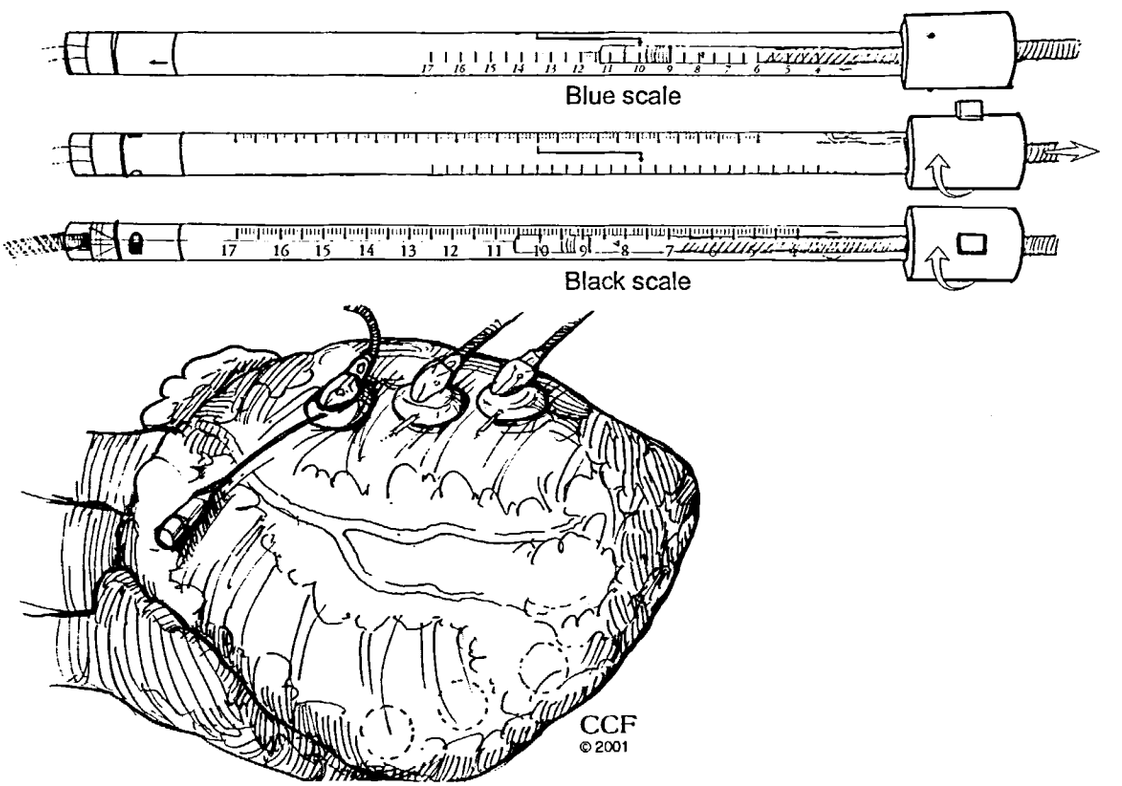


7 The tension member has a leader segment on one end and a fixed pad on the other end. The leader segment of the tension member is inserted into the distal end of the needle tube and fed through the length of the needle tube. Once the tension member leader exits through the needle tube, the needle tube is removed, leaving the tension member traversing the ventricular walls and chambers. The tension member is pulled through the heart until the pad rests against the RV wall (see inset).

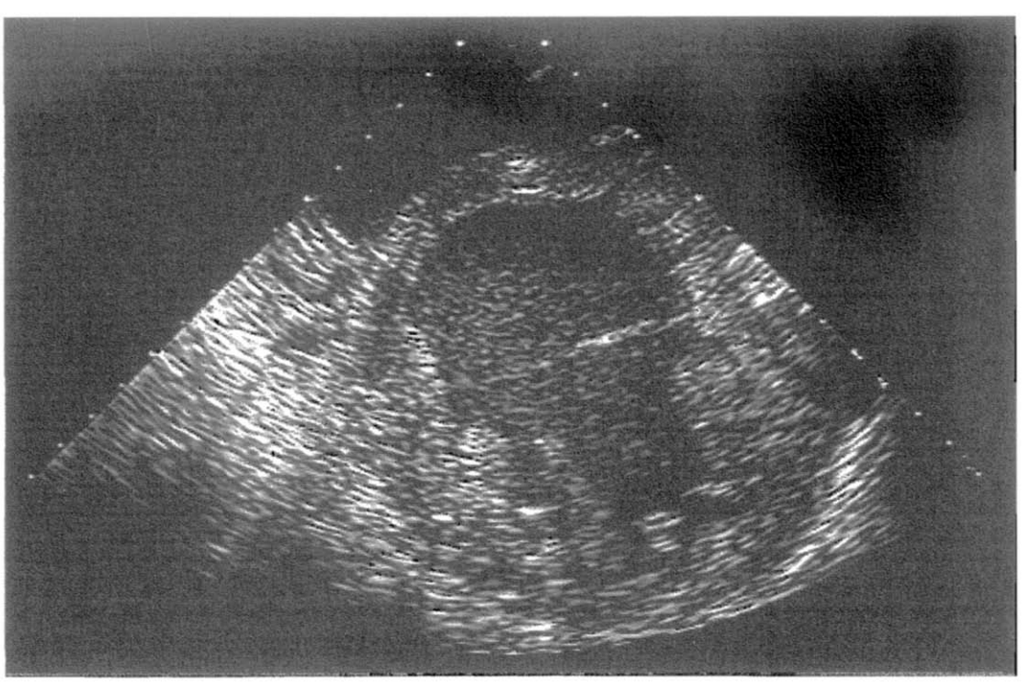
8 After placement, short-axis EE confirms tension member positioning and chamber bisection. If tension member placement is not acceptable, then the tension member is removed and repositioned. Acceptable placement is judged by avoidance of major coronary vessels, papillary muscles, and valvular structures and by appropriate bisection of the LV.



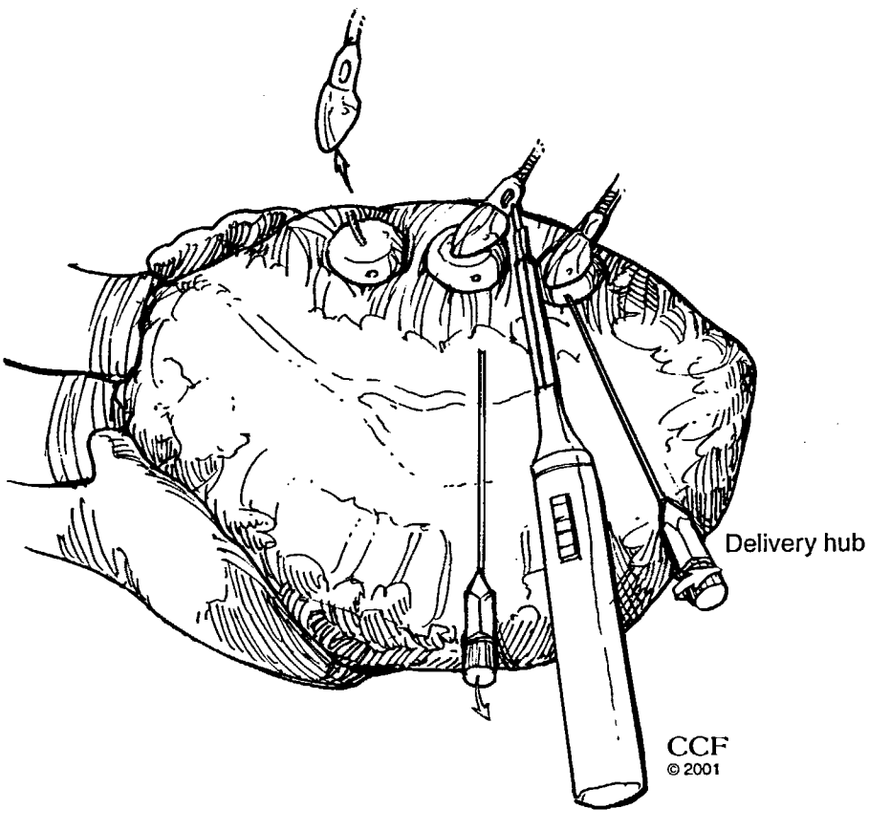
9 A deployable pad and a measurement and tightening (M/T) device are placed over the end of the tension member. The M/T device is advanced until both pads rest on the outside of the heart (see inset). The M/T device is adjusted until any potential distance between the RV wall and septum is collapsed (as can be seen on the EE) without changing the LV geometry. In this position, the M/T device measures the pretightening LV dimension by reading the position of the green index mark on the primary (black) scale.



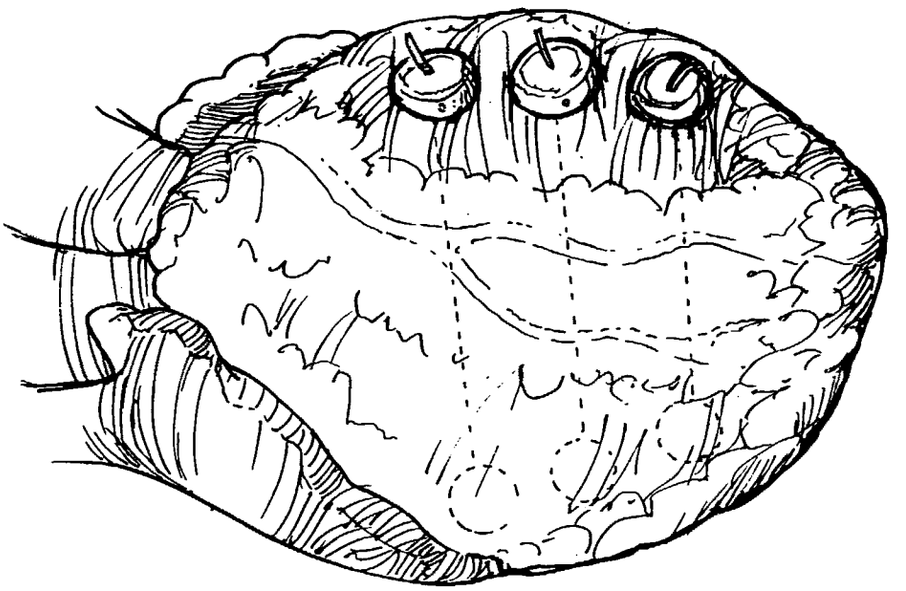
10 The process for tension member insertion is repeated for the middle and apical tension members. Starting with the basal tension member, the M/T device is tightened to apply the 20% stress reduction. The green index mark is adjusted to the same value on the second (blue) scale as is measured on the black scale. Fine and coarse adjustment when using the M/T device and handling the black plunger enables large size adjustments initially and then finer tuning for precise measurement. The tightening procedure is repeated for all tension members.



11 After all three tension members are tightened, EE is reperformed to confirm the shape change and to evaluate mitral and tricuspid valve regurgitation.



12 To lock the deployable pad in place, the delivery hub of the deployable pad is rotated clockwise until two clicks are heard. Any excess tension member is trimmed with a cautery device (an electric "hot knife") through the trimming window of the M/T device.



13 On completion of the Myosplint implantation procedure, the heart has been successfully reshaped by tightening the three tension members.

ered devices can become outpatients and achieve an acceptable quality of life. Myosplint implantation is not recommended for such severely ill, end-stage heart failure patients.

In patients with dilated cardiomyopathy, functional mitral regurgitation often results from annular dilatation and ventricular enlargement. Bolling and colleagues[6] showed that patients with severe ventricular dysfunction and severe mitral regurgitation improve clinically after mitral valve repair alone and reported actual 1- and 2-year survival rates of 82% and 71%, respectively. In our experience in 44 patients with mitral regurgitation and an LV ejection fraction below 35% who underwent isolated mitral repair ($n = 35$) or replacement ($n = 9$), the 1-, 2-, and 5-year survival rates were 89%, 86%, and 67%, respectively, with an improvement in NYHA class from 2.8 ± 0.8 preoperatively to 1.2 ± 0.5 at follow-up.[7] The Myosplint implantation can be performed in combination with mitral valve repair for patients with dilated cardiomyopathy and mitral regurgitation.

PLV reduces wall stress and improves systolic function by resecting a large part of the LV muscle. However, PLV is a major surgical procedure requiring cardiopulmonary bypass and loss of a large segment of otherwise viable myocardium; the procedure may also worsen preexisting diastolic dysfunction.[8] In our experience in 62 patients at the Cleveland Clinic, we observed a significant early failure rate, with 60% 3-year survival and 26% freedom from failure.[9] Although improvements in subjective clinical status and objective measurements (maximum exercise oxygen consumption) are observed in some patients, PLV currently is not considered to be a predictable, reliable alternative to transplantation.

The Myosplint has several possible advantages. It can be easily implanted on a beating heart; reduces wall stress without resecting otherwise viable muscle; improves systolic function without creating major changes in diastolic function; and does not require a power source.

A newly designed C-device is being evaluated clinically to further improve the implantation procedure. This new C-device has two separate arms, one arm with a guide tube and another arm with a hard stop (Fig VA). Each arm is adjusted to the entry or exit point independently, and a vacuum is applied to ensure the attachment of each arm onto the epicardial surface. The two arms are then connected by aligning the handles. The two handles are snapped together to lock in place (Fig VB). The handle can then be used to stabilize the C-device during the delivery of the needle assembly through the chamber. With the current one-piece C-device, one end sometimes moves as the surgeon adjusts the other end. We anticipate that the new C-device will be used in future Myosplint implantation surgeries.

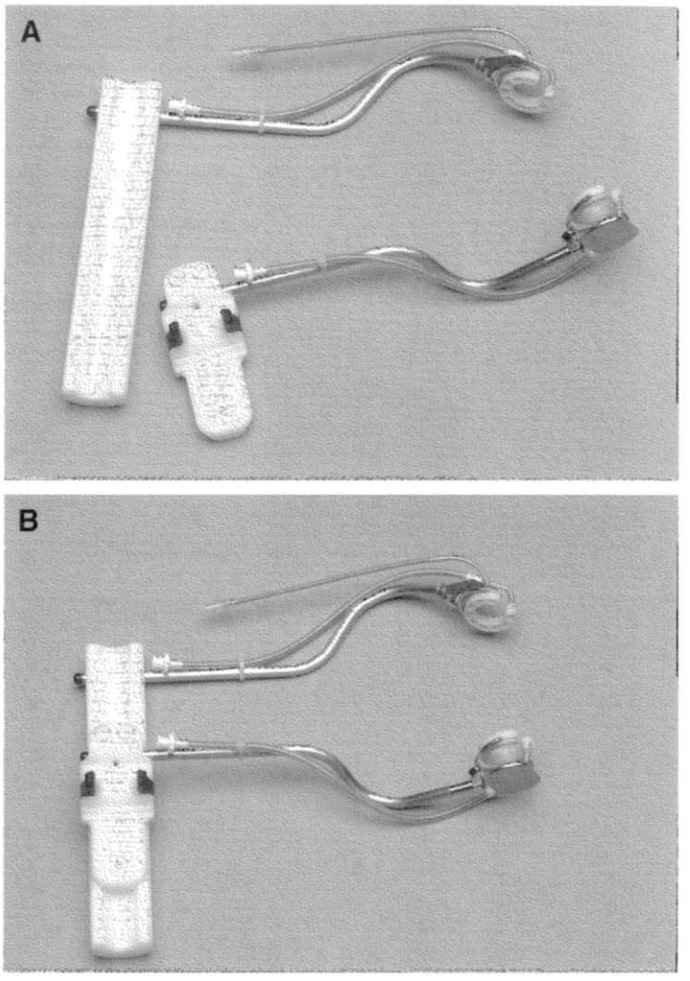


Fig V. The new C-device has two separate arms to allow independent adjustment of the position of each end.

The Myosplint is a passive device for treating dilated cardiomyopathy and is easily applied on a beating heart. The device is now being evaluated clinically in Germany and the United States as an adjunct for those needing valve surgery or as the sole surgical therapy for patients with idiopathic cardiomyopathy.

REFERENCES

1. McCarthy PM, Takagaki M, Ochiai Y, et al: Device-based change in left ventricular shape: A new concept for the treatment of dilated cardiomyopathy. J Thorac Cardiovasc Surg 122:482-490, 2001
2. Fukamachi K, McCarthy PM, Takagaki M, et al: Myosplint improved left ventricular function by improving systolic elastance and maintaining diastolic compliance in a canine cardiomyopathy model [abstract]: Circulation 104:II-410, 2001
3. Konertz WF, Shapland JE, Hotz H, et al: Passive containment and reverse remodeling by a novel textile cardiac support device. Circulation 104 (suppl I):I-270-I-275, 2001

4. Melvin DB: Ventricular radius reduction without resection: A computational analysis. ASAIO J 45:160-165, 1999
5. McCarthy PM, Smedira NO, Vargo RL, et al: One hundred patients with the HeartMate left ventricular assist device: Evolving concepts and technology. J Thorac Cardiovasc Surg 115:901-912, 1998
6. Bolling SF, Pagani FD, Deeb GM, et al: Intermediate-term outcome of mitral reconstruction in cardiomyopathy. J Thorac Cardiovasc Surg 115:381-388, 1998
7. Bishay ES, McCarthy PM, Cosgrove DM, et al: Mitral valve surgery in patients with severe left ventricular dysfunction. Eur J Cardiothorac Surg 17:213-221, 2000
8. Artrip JH, Oz MC, Burkhoff D: Left ventricular volume reduction surgery for heart failure: A physiologic perspective. J Thorac Cardiovasc Surg 122:775-782, 2001
9. Franco-Cereceda A, McCarthy PM, Blackstone EH, et al: Partial left ventriculectomy for dilated cardiomyopathy: Is this an alternative to transplantation? J Thorac Cardiovasc Surg 121:879-893, 2001

From the Department of Biomedical Engineering, Lerner Research Institute, Department of Thoracic and Cardiovascular Surgery, Kaufman Center for Heart Failure, and Department of Medical Illustration, The Cleveland Clinic Foundation, Cleveland, OH.

Address reprints requests to Patrick M. McCarthy, MD, Department of Thoracic and Cardiovascular Surgery/F25, Kaufman Center for Heart Failure, The Cleveland Clinic Foundation, 9500 Euclid Ave., Cleveland, OH 44195.

1522-2942/02/0702-0001$35.00/0
doi:10.1053/otct.2002.30889